UNDERSTANDING AND MANAGING ACUTE DIARRHOEA IN INFANTS AND YOUNG CHILDREN

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FOREWORD

In commending Dr. O.P. Ghai's brief yet exhaustive elucidation of the science and art of managing childhood diarrhoea, I would like to underline the unusual social priority deserved by this fairly simple procedure. Perhaps the principles involved are in a sense so elementary that it takes an effort on the part of the professional to adopt it in practice with a seriousness appropriate to its validity and usefulness.

According to one estimate, there are 1400 million episodes each year of young child diarrhoea and 5 million consequent deaths in the developing countries. While the incidence of diarrhoea is understandable in an environment where safe water is not always available and sanitary practices are a frequent casualty, the recurring massive toll of lives and health, especially of children, is entirely unnecessary. The central scientific message of Dr. Ghai's paper is that the tragedy is preventable at affordable cost by simple methods within the means of the generality of the people. I would add the good news that the preventive effort has begun, successfully and on a social scale, across the developing world, including here in India.

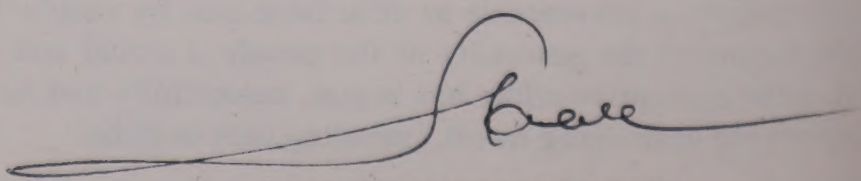
A major condition has to be fulfilled before childhood deaths from diarrhoea become a thing of the past. In one word, it is building of awareness in the minds of the people, including health workers at different levels, about the consequents of diarrhoea to the individual child and to society as a whole, and secondly about the means available and accessible for controlling them.

What are the consequences? These go even beyond the nipping of large numbers of young lives. In collusion with other adverse factors like malnutrition and vaccine-preventable childhood diseases, recurrent

diarrhoea reduces absorption of food as well as of lessons at school, retards growth, lowers the quality of life, increases the burden on the health system, puts a strain on the public health budget and acts as a disincentive to limiting family size. Its overall effect on society—adults as well as children—is to depress social and economic productivity.

The principle of oral rehydration technology to prevent severe dehydration and death has been known for a long time. Indeed, it is in one form or another part of the traditional art of healing in most parts of the world including India. It is another matter if this practice has gone into disuse, largely from the impact of certain commercial aspects of latter-day medical practice. The scientific validation and general acceptance of the oral rehydration therapy has therefore come as a welcome development of recent times. So, too, is the parallel understanding of the extent and severity of diarrhoeal dehydration as a result of investigations over the past few years. The time has thus come to mount a decisive assault on a hitherto under-rated public health problem—by transferring the knowledge, skills and confidence to mothers and families even in remote villages for intervening on their own initiative whenever diarrhoea occurs.

This educational process of communications has to spread in ripples starting from the medical scientist, moving through the practising professional and the front-line health worker, to reach the mother in the household. It is an encouraging sign of our transitional times that necessary trend by making professional knowledge intelligible and available to a widening and increasing concerned audience.



(S. KRISHNAKUMAR)

CONTENTS

UNDERSTANDING AND MANAGING ACUTE DIARRHOEA IN INFANTS AND YOUNG CHILDREN

SYNOPSIS

Acute diarrhoea is a major cause of morbidity and mortality in infants and young children all over the world, more so in the developing countries. Encouragement of breast feeding, better food hygiene, improvement of nutritional status of children and good environmental sanitation are important strategies for lowering the incidence of diarrhoea. It is equally important to reduce the high rate of diarrhoeal deaths because of loss of fluid and electrolytes from the body as well as malnutrition and its sequelae due to repeated attacks of acute diarrhoea. The principal interventions to achieve these objectives fall into the following categories in descending order of priority.

1. Fluid and Electrolyte Management

Mother should be encouraged to feed the baby with simple home-available or home-made fluids from the very onset of the illness, in order to quickly replace the fluids lost in diarrhoeal stools. This prevents dehydration (loss of water from the body) and maintains circulatory and renal functions. This should be followed by a clinical back up for more severe cases, who might require therapy with an oral rehydration solution with approved constituents. When fluid replacement is started early, intravenous medication is seldom necessary.

2. Nutritional Management

Oral rehydration therapy prevents deaths due to dehydration, but by itself is not enough to provide long-term health benefits and reduce the overall infant and child mortality. Children with recurrent attacks of diarrhoea become undernourished. Early feeding hastens recovery. Infants should continue to be fed as was done prior to the onset of diarrhoea, breast-feeding should not be interrupted and refeeding should be started early. During convalescence extra nutrient-rich food should be given to ensure rapid and complete nutritional recovery.

3. Treatment of Causative Agent

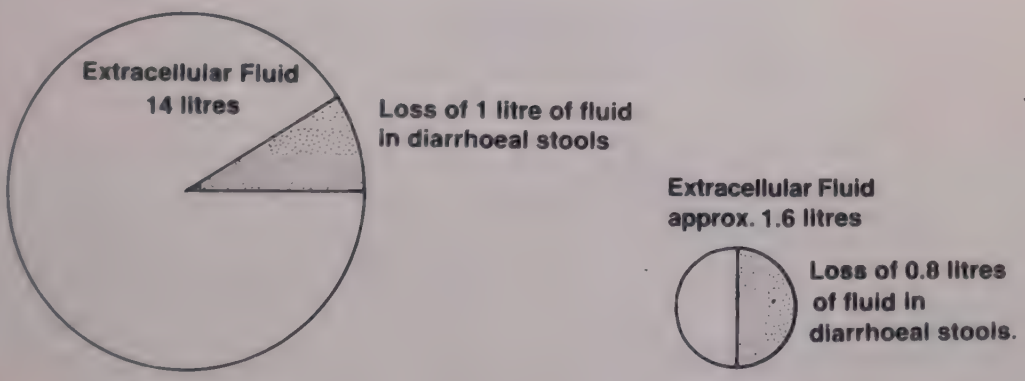
A vast majority of diarrhoeal episodes are caused by viruses or agents which do not require antimicrobial treatment. Antimicrobial treatment is needed only for dysenteries, cholera and infections with bacteria and protozoa which invade the mucosa. These account for less than 10 per cent of cases. Injudicious use of antimicrobial agents causes emergence of resistant strains of organisms, resident flora of the gut which are protective are destroyed and toxic complications of some antibiotics are a real and significant danger.

4. Other Drugs

Drugs such as antimotility agents, antisecretory drugs, binding agents and other time-honoured medications for diarrhoea do not offer any significant benefits when subjected to scientific scrutiny. In fact, these could be potentially harmful. Often these divert attention and resources from the central issues of fluid therapy, nutritional management and specific treatment when required. Antimotility drugs are potentially dangerous and must not be used for acute diarrhoea in infants and children below 5 years of age.

I. INTRODUCTION

Diarrhoea is a common but potentially serious illness in early childhood. A child suffers, on an average, 10 to 15 episodes of diarrhoea in the first five years of life. Of these, three to five occur in the first year of life. A child may lose almost as much water and electrolytes from the body during an episode of diarrhoea as an adult, since the length and surface area of intestinal mucosa of a child, from where the diarrhoeal fluids are secreted, are fairly large. Loss of one litre of fluid from the body of a child weighing 7 Kg is much more hazardous compared with a similar depletion from an adult of 70 Kg weight. Significant dehydration disturbing the balance of electrolytes and acid-base status of the body occurs in about 2 to 3 per cent of all cases of diarrhoea. Some of these cases may prove fatal, if fluids and electrolytes are not replaced to restore normal circulation and body functions which are impaired in the dehydrated state.



Loss of one litre of fluid in diarrhoeal stools in adults (70 Kg) constitutes about 7 per cent of total extracellular fluid compartment. Loss of 0.8 litres of fluid in an infant (7 Kg.) with approximately 1.4 to 1.6 litre of extracellular fluid (ECF) volume is equivalent to almost half of the latter, and therefore it is much more hazardous.

Fig. 1

Diarrhoea has been shown to have significant impact on nutrition. Most field studies identify diarrhoea as the major determining factor leading to malnutrition in the developing countries. It is the child with multiple episodes of diarrhoea and particularly chronic diarrhoea, who suffers most severely from protein-energy malnutrition. But even a brief episode of diarrhoea leads to the loss of 1 to 2 per cent of body weight per day. Infants and young children in developing countries are sick for about 10 per cent of the time (or nearly 30 days per year) with diarrhoeal illness. Thus over the time even the creeping deficit associated with mild illness can accumulate to become a major nutritional deficiency. If diarrhoea becomes unusually prolonged or is recurrent, the child becomes severely malnourished, since he/she also loses nutrients through stools. The appetite is impaired and food is often withheld from the child by the mother due to an erroneous belief that starvation rests the bowel and promotes early recovery from diarrhoea.

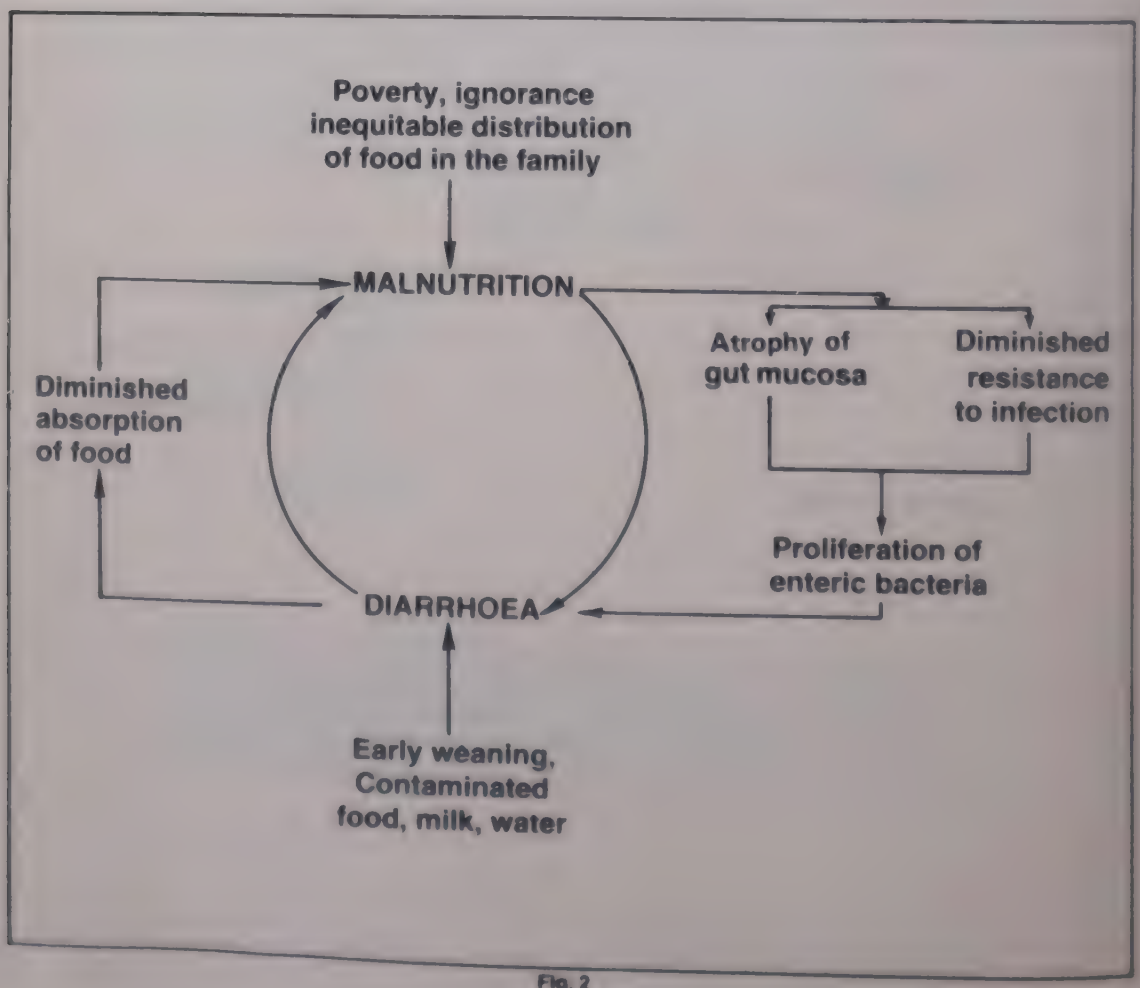


Fig. 2

Atrophy of the intestinal epithelium in cases of malnutrition causes malabsorption and accentuates malnutrition. A vicious cycle of diarrhoea-malnutrition-diarrhoea sets in. Since, malnourished children are more prone to suffer from other infections as well, diarrhoea-malnutrition symptom complex contributes to the large majority of early childhood deaths either directly or indirectly. Prevention and proper management of diarrhoea,therefore, assumes a high priority.

II. WHAT CAUSES DIARRHOEA

Most cases of acute diarrhoea in young children are due to infections with a wide variety of organisms. All of these are not amenable to the presently available antimicrobial agents. The exact incidence of these microbes may vary from place to place and at different periods of the year. Broadly speaking, 30 to 40 per cent of diarrhoeal episodes are caused by viruses, of which rotavirus is the best example. About 50 per cent are due to bacterial infections of the gut. The presence of bacteria in the stools by itself is not proof of these being causative agents. Bacteria cause diarrhoea by two distinct mechanisms viz.

(i) through the action of toxin and
(ii) direct invasion of the intestinal mucosa.

Cholera vibrio is an important agent causing diarrhoea in some parts of India and also when normal sanitary arrangements are overstretched during major religious congregations such as Kumbh melas and during natural calamities. In young children, 30 to 40 per cent of all cases of diarrhoea can be attributed to toxin producing strains of Escherichia coli. These as well as cholera vibrio do not actually invade the intestinal mucosa. There is usually no evidence of inflammatory response in the gut. Toxins produced by these organisms stimulate the enzyme *adenyl cyclase* in the enterocytes, which increases the production of cyclic AMP. The latter promotes active losses of electrolytes and water from the intestinal cells. There are additional mechanisms of diarrhoea due to cholera vibrio.

In 5 to 10 per cent of all cases of diarrhoea the microbes such as shigella actually invade the mucosal cells, proliferate there and cause necrosis. The damaged cells are shed resulting in passage of blood and mucus in the stools, the clinical syndrome of dysentery. Infection with this organism needs to be managed with antimicrobial agents.

RECOGNISED ENTEROPATHOGENS FOR ACUTE DIARRHOEA

Viruses	Bacteria	Parasites
Rotavirus Norwalk Agent Adenoviruses Calicivirus Coronavirus Astrovirus	Escherichia coli i) Enterotoxigenic* ii) Enteropathogenic* iii) Enteroinvasive iv) Enterohaemorrhagic* v) Enteroadherent Vibrio cholerae* Shigella* Campylobacter jejuni Staphylococcus aureus* Clostridium perfringens* Clostridium difficile* Non-typhoid salmonella Yersinia enterocolitica Vibrio parahaemolytica Aeromonas hydrophila Bacillus cereus*	Entamoeba histolytica Giardia lamblia Strongyloides stercoralis Trichuris trichuria Cryptosporidia

*These organisms produce enterotoxins

Table 1

Campylobacter jejuni invades the intestinal mucosa and is isolated in 5 to 10 per cent of diarrhoeal cases. Non-typhoid salmonella are an infrequent cause of diarrhoea especially in immune-compromised infants. It may cause severe morbidity and may end fatally due to systemic infection.

Infestations with Entamoeba histolytica and Giardia lambia result in subacute and chronic illness. Malaria due to Plasmodium falciparum is often implicated as a cause of acute diarrhoea in tropical countries. Intestinal helminths are not causes of acute diarrhoea. Their frequent presence in stools simply reflects high prevalence in the population.

MECHANISM OF ACTION OF ENTEROPATHOGENS	
Organisms which adhere to the mucosa and produce enterotoxins (secretory diarrhoea, no inflammation of the gut).	Enterotoxigenic E. coli Vibrio cholerae
Organisms which damage the brush border and its enzymes (cause carbohydrate malabsorption).	Enteropathogenic E. coli (some of these are enteroadherent) Rotavirus
Organisms that invade the mucosa and proliferate in the intestinal epithelium.	Shigella Enteroinvasive E. coli
Organisms which proliferate in the lamina propria and invade the mesenteric lymph anodes.	Non-typhoid salmonella; Campylobacter jejuni; Yersinia enterocolitica
Disordered small intestinal epithelial renewal.	Rotavirus

Table 2

III. HOW DOES DIARRHOEA CAUSE SIGNIFICANT PHYSIOLOGICAL DISTURBANCES IN THE BODY

Water constitutes about 75 per cent of body weight at birth and approximately 60 per cent of child's body weight is present in two separate compartments—the extracellular (E.C.F.) and intracellular (I.C.F.) The extracellular compartment includes circulating blood, interstitial fluid and secretions. Diarrhoea losses come from ECF and replacement fluids should be of a similar composition: relatively rich in

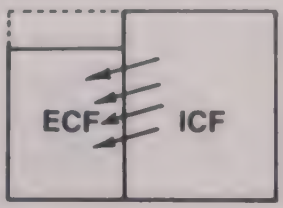
sodium with low potassium. Kidneys regulate the electrolyte content of the extracellular compartment by filtering, concentrating, diluting and reabsorbing fluids and metabolites from the circulation. Functional ability of the kidney of very young infants is not fully developed as compared with older children.

Large amounts of water and water-soluble nutritive substances such as electrolytes, metabolites and vitamins are lost from the body during diarrhoeal episodes. Loss of water from the body causes a reduction or shrinkage in the volume of extracellular compartment. In about half of these cases, the concentration of sodium in the plasma or extracellular compartment remains nearly normal (about 140 mEq/L). Since excessive sodium may be lost in the stools in another 40 to 45 per cent of cases, there is a relative decline in the serum and ECF sodium level (hyponatremia). Sodium is a major osmotic determinant of ECF Therefore, the osmolality of ECF falls, causing movement of water from the extracellular to intracellular compartment. This causes further shrinkage of the already reduced extracellular compartment volume. Skin turgor or elasticity is normally maintained by the presence of water and fat in the tissues. Shrinkage of extracellular water in both hypo and isonatremic types of dehydration impairs the skin elasticity. The skin appears to be wrinkled like that of an old man. On pinching, it takes a few seconds for the skin folds to return to normal.

In about 5 per cent of diarrhoea cases (especially if the mother has given fluids with more salt) serum sodium levels may be elevated to more than 150 mEq/L. In these patients, the osmotic pressure of ECF is relatively higher. Therefore, water moves from inside the cells to the extracellular compartment. This restores the depleted extracellular fluid and therefore partially masks the loss of skin turgor. The skin may appear soggy or leathery. The physician is likely to erroneously underestimate a severe case of hypernatremic dehydration as mild dehydration unless he/she takes into account the other more important sequelae of dehydration such as circulatory or renal impairment.

As the extracellular compartment is depleted, the blood volume is reduced. This results in a weak thready pulse and a fall in blood pressure. Extremities appear cold. Because of low hydrostatic pressure in the renal glomeruli, the filtration of urine is reduced. The quantity and frequency of urination falls. This is ominous because poorly functioning kidneys cannot regulate the metabolic derangements. Urine flow is a good indicator of the severity of illness. In severe cases, renal failure may eventually set in.

Fluid moves from ECF to ICF compartment in hyponatremic dehydration accentuating fluid depletion in ECF



Fluid moves from ICF to ECF compartment in hypernatremic (hypertonic) dehydration, thus partially compensating for fluid depletion of ECF compartment

Fig. 3

Diarrhoeal stools contain large amounts of potassium. Therefore, the serum potassium level invariably falls if diarrhoea persists for more than a few days. The affected children present with abdominal distension and hypotonia of muscles. Electrocardiogram shows ST depression and flat T waves.

Since intestinal secretions are alkaline and considerable bicarbonate is lost in diarrhoeal stools, acidemia usually accompanies diarrhoeal dehydration. Patients in such cases remain asymptomatic till the base excess falls to 12 mMol/L. As the base excess falls below this level, the breathing becomes deep and rapid (Kussmaul breathing).

To sum up, in early and mild cases of diarrhoea, the child may be thirsty and slightly irritable. As the diarrhoea continues and dehydration worsens, the child becomes more irritable and develops a pinched look. His/her fontanelle, if open, is depressed, the eyes appear sunken, the nose is pinched, and the tongue and the inner side of cheeks appear dry. Abdomen becomes distended in hypokalemia. The child passes urine at longer intervals. As acidosis worsens, the breathing becomes deep and rapid. In extreme cases, the child appears moribund, pulse appears to be weak and thready, blood pressure falls and the quantity of urine passed is markedly reduced. Children with severe dehydration succumb rapidly if they are not promptly treated.

CLINICAL FEATURES OF DEHYDRATION

Mild	Moderate	Severe
Irritable	Irritable	Moribund, apathetic
	Weak pulse	Peripheral circulatory failure (cold extremities, warm body, excessive blanching, weak pulse)
	Some reduction in urine volume	Marked reduction in urine volume
	Fontanelle depressed	Fontanelle markedly depressed
	Eyeballs sunken	Eyeballs markedly sunken
	Facies dry and pinched	Facies markedly dry and pinched
	Bucccal mucosa dry	Bucccal mucosa dry
	Lips parched	Lips parched
	Loss of skin turgor (except in hypernatremic variety)	Loss of skin turgor (except in hypernatremic variety in which it may not be prominent)
Thirsty	Thirsty	Thirsty

Table 3

IV. CLINICAL APPROACH TO DIAGNOSIS

In *rotavirus diarrhoea*, vomiting is an early feature and diarrhoea is more severe. Most patients have mild to moderate fever. Norwalk virus infection occurs in slightly older infants and pre-school children.

Stools are large and watery in secretory diarrhoea due to infection with toxigenic strains of *E. coli* or *cholera vibrio.* The foecal matter appears as curdy deposits. Vomiting is common in cases of cholera.

Fever, abdominal cramps and tenesmus with passing of blood and mucus in stools indicate *dysentery* (colitis), due to infection with *shigella.*

Small amounts of blood in stools may be seen in infections with *salmonella, campylobacter or invasive strains of E. coli.* Infection of the gut with staphylococci, *Candida albicans or Clostridium difficile* should be suspected in severe cases of diarrhoea in very sick infants, who had received prolonged treatment with broad spectrum antibiotics.

V. LABORATORY APPROACH TO DIAGNOSIS

The following investigation may be carried out in order of priority, but these are not absolutely essential for effective management of the case, which can be done equally well on the clinical basis.

(i) Microscopic examination of stools for pus cells, red blood cells and macrophages (cellular exudate) and presence of cysts or vegetative forms of Entamoeba histolytica or Giardia lamblia.

(ii) Blood investigations such as pH, base excess, electrolytes such as N^+,K^+ haemoglobin level, urea and osmolality.

(iii) Record of the pH or reaction of the stools by dipping a strip of pH indicator paper of blue litmus paper in the stools suspension.

(iv) Culture of stools for enteropathogenic bacteria.

(v) Tests for the presence of toxins in the organisms cultured from stools e.g. distension of the isolated rabbit ileal loop or GM_1 ELISA etc.

(vi) Tests for the presence of rotavirus by electron microscopic examination or by ELISA test.

VI. WHAT IS THE PHYSIOLOGICAL BASIS FOR MANAGEMENT

In most cases of acute diarrhoea, electrolytes such as chloride and sodium besides water are actively secreted from the gut mucosa and thus lost in stools. However, physiologists observed that while water and sodium were being lost, nutrients such as glucose, aminoacids and dipeptides continued to be absorbed without difficulty in a majority of cases. The uptake of glucose and other nutrients by the body is an enzyme mediated active physiological process. The carrier mechanisms for the transport of glucose and that for sodium across the cell membrane are interlinked. As glucose is absorbed in the gut, sodium is carried along and also absorbed, even though it is being actively lost in secretory diarrhoeas due to the effects of the toxin elaborated by some causative agents such as enterotoxigenic strains of Escherichia coli and cholera vibrio. If an iso-osmolar solution of glucose and sodium is given orally, glucose and sodium are absorbed. *Sodium absorption also promotes absorption of water This is the physiological basis of oral-rehydration therapy,* which is a fascinating advance of modern medicine and has probably saved more lives than any other treatment modality.

Oral rehydration solution with a concentration of 90 mEq/L of sodium is ordinarily used for all types of diarrhoea at all ages. There may be a minimal risk of hypernatremia developing in the neonates and very young infants with immature kidney functions. However, the risk is very small and it can be easily eliminated if one part of water is given after two parts of ORS. After hydration has been achieved, ORS with a lower concentration of sodium (e.g. 60 mEq/L) will suffice for maintenance needs of fluid and electrolytes. Alternatively the WHO formula can be supplemented with drinks of water or breast milk. *The fear of hypernatremia is often exaggerated.* In fact, *hypernatremia should be corrected slowly.* A sudden fall of sodium concentration with the use of very hyponatremic solutions orally may cause sudden changes in movements of fluids across cell membrane in neurones and precipitate convulsions.

Oral rehydration solution (ORS) should preferably be given with a teaspoon or consumed in small sips from a cup or tumbler. A child with profuse vomiting is more likely to retain the fluid if it is consumed in small sips. Large gulps of fluid stimulate gastrocolic reflex resulting in a quick passage of motion and often vomiting.

VII. PRINCIPLES OF THERAPY

1. SEVERE CASES

The status of dehydration should be determined quickly and emergency treatment instituted if necessary.

2. MILD TO MODERATE CASES

(i) Fluid, electrolyte and acid-base homeostasis should be preserved and maintained.

(ii) Nutritional status of the patient should be restored as early as possible. Breast feeding should be continued. Refeeding should be started early and extra food supplements should be given during convalescence.

(iii) Antimicrobial agents should be sparingly used and only for specific indications such as dysentery and cholera.

(iv) There is no scientific basis for the use of anti-motility or binding agents.

(v) Associated features such as persistent vomiting, abdominal distension and convulsions should be managed appropriately.

3. FLUID MANAGEMENT

The key to effective fluid management in childhood diarrhoea is early replacement of fluid losses, starting with the first sign of liquid stool. Plenty of fluid should be given to the child early in the illness to prevent dehydration. As long as renal function is maintained, profound electrolyte and pH disturbances do not occur.

(i) Initial Management with any Fluid available at Home

The mother should be advised to offer fluids that are easily available at home in as much quantity as the child can take orally without vomiting. Thus coconut water, butter-milk, rice kanji with salt, lemon-sugar-salt beverage (salted-nimbupani-sherbat), weak tea etc. may be given *ad lib* either with a teaspoon or in small sips from a tumbler. In mild cases, diarrhoea and vomiting are generally controlled within a short period and dehydration does not develop.

(ii) Oral Rehydration Therapy (ORT) with Home-made Solution

A fairly satisfactory solution for oral rehydration can be prepared at home by mixing eight level teaspoonfuls of cane sugar (40 grams of sucrose), one level teaspoonful of table salt (five grams of NaCl) with or without a lemon squeezed in one litre of potable water. Since 2 g of sugar releases 1 g of glucose, 40 g of sucrose is used. Alternatively a 3 finger pinch (upto the first crease) of table salt and closed fistful of cane sugar are mixed in half a litre of water.

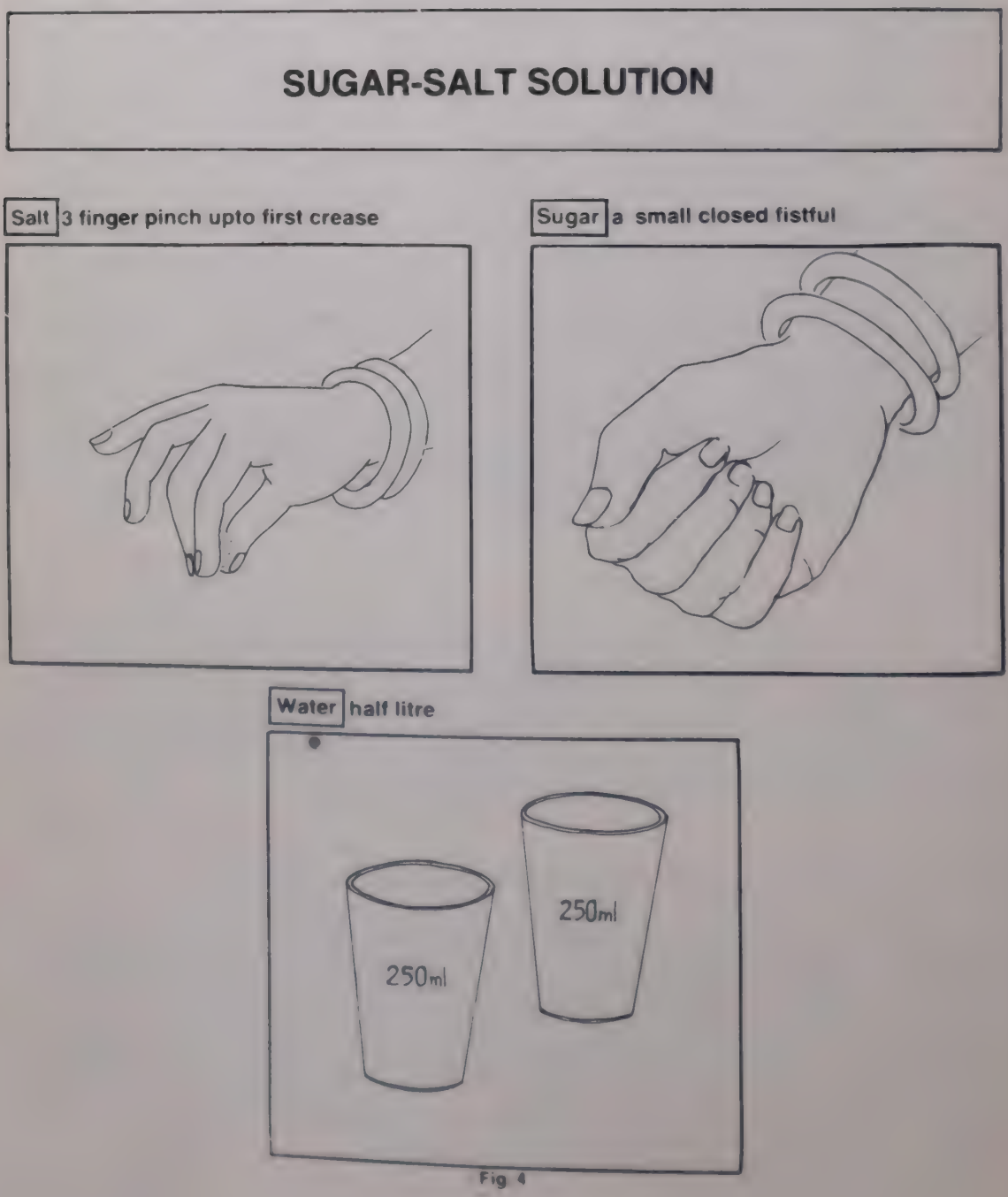


Fig 4

Some health workers have successfully substituted 20 g of glucose or 40 g of cane sugar by 50 g of puffed rice powder in the home made ORS and got equally satisfactory results. It may be possible to replace a part of puffed rice powder with other cereals or cooked legumes, such as rice and dal gruel or khichri in areas where it is a culturally preferred mode of diarrhoeal management. It takes about 5 to 7 minutes of boiling to prepare this. The preparation must be allowed to cool and dissolved properly before administration.

(iii) Oral Rehydration Salts (ORS) Solution

If the diarrhoea is prolonged and dehydration becomes evident, it is desirable to rehydrate the child orally by administering a solution with the composition approved by the World Health Organization (glucose 20 g; sodium chloride 3.5 g; trisodium citrate 2.9 g or sodium bicarbonate 2.5 g and potassium chloride 1.5 g dissolved in one litre of safe drinking water). This solution provides 90 mEq/L of Na, 20mEq/L of K, 80 mEq of Cl and 30 mEq/L of HCO_3. It is administered in small sips or with a teaspoon to prevent vomiting or rapid passage of stools due to hyperactive gastro-colic reflex. It should be given freely till the dehyration is corrected. *Broadly speaking a one-year-old infant needs about 1000 ml of ORS in 24 hours. To compensate for additional losses with continuing diarrhoea, an extra 100 ml of oral rehydration solution should be given for each diarrhoeal stool.* The quantity of ORS required for 6 to 12 hours may be prepared at a time to avoid extraneous contamination of ORS during storage.

4. EMERGENCY TREATMENT

Severe dehydration is a major paediatric emergency, which may end fatally if not managed early and adequately. A severely dehydrated child appears *drowsy,* is *apathetic* and becomes moribund. As he/she goes into *peripheral circulatory* failure, extremities appear cold, though the body may be warm. The skin blanches for more than a few seconds. *Pulse is weak and fast.* The quantity of urine passed is reduced or the child does not urinate for several hours. Besides these, other clinical features more characteristic of moderate dehydration are also present. These include *depressed anterior fontanelle (if open), sunken eyeballs, dry oral mucosa and poor skin turgor.* They are thirsty and irritable. In children with nutritional wasting of the subcutaneous tissue, the skin elasticity is impaired even when there is no dehydration. Therefore, in malnourished children, associated dehydration should be diagnosed if the little finger of the examiner, passed over the inner side of the cheek, feels dry.

Less than one per cent of all cases of diarrhoea in the community develop severe dehydration and circulatory or renal insufficiency and need emergency treatment to save their life or prevent irreversible physiologic damage. Therefore, *it is not necessary to give intravenous fluids in any but the most severe case of diarrhoea.* Besides being expensive and time consuming, intravenous medication may be potentially hazardous because of *the risk of overhydration and possibility of introducing infection in the veins causing thrombophlebitis or septicaemia.*

If a child develops severe dehydration and/or has persistent vomiting with or without marked upper abdominal distension, it is prudent to start an intravenous drip of Ringer's lactate solution, given at a rate of 30 ml/kg/ of body weight in the first hour. The rate of drip is slowed to 20ml/kg/hr in the next 2 hours. If the child does not pass urine within three hours of starting intravenous infusion, acute renal failure should be suspected. It is important to check all signs to be sure that the child is fully hydrated before labelling it as acute renal failure. In cases of acute renal failure, it is not desirable to further push fluids and specialist's help should be sought for the management of renal failure. A child who starts passing urine in two hours should receive 40 ml/kg of Ringer's lactate solution intravenous in the next two hours as well. *Concurrently, oral rehydration therapy should also be started as described above.* The patient is kept under surveillance and as his condition improves and he starts taking ORS, the intravenous medication can be discontinued. It should be possible to remove the I.V. needle in almost all cases within four hours as rehydration will be completed by then. Thereafter oral rehydration therapy will suffice for replacing continued losses and maintenance requirements of fluids.

VIII. NUTRITIONAL MANAGEMENT OF DIARRHOEA

A considerable quantity of nutrients is lost in diarrhoeal stools. Appetite is impaired and food is often withheld from the child by the mother because of an erroneous belief that rest to the bowel promotes early recovery. Some malabsorption of food occurs during diarrhoea, but it is only partial. *Most nutrients are well absorbed during diarrhoea.* Some hydrolytic (disaccharidases) enzymes and absorptive mechanisms for glucose and aminoacids may be partially compromised during viral diarrhoea. Transient carbohydrate malabsorption may occur. Since carbohydrates may pass unchanged in the lower gut, these raise intraluminal osmotic pressure and draw water from the gut by osmosis

and increase the severity of diarrhoea. Unabsorbed carbohydrates are also metabolised to short-chain fatty acids by colonic bacteria and are then absorbed from the colon. These pathological changes are transient and do not last for more than a few days in most cases. Therefore it is safe and desirable to continue feeding in acute diarrhoea.

Since children with diarrhoea develop protein-energy-malnutrition, the diet should be easily digestible and nutritionally balanced. Presence of nutrients in the gut promotes absorption of sodium and water and hastens recovery of the intestinal epithelium because food in the intestine stimulates rapid cell turn-over and renewal of intestinal lining. *More lives are lost because of unnecessary starvation in diarrhoea.*

Feeding during diarrhoea may be considered under three heads:

(a) Exclusively breast-fed infants;

(b) Fully weaned infants receiving only animal milk;

(c) Infants who receive both solid and liquid diet whether partially breast-fed or not.

1. Breast-fed babies: The infant should continue to be breast-fed during an attack of diarrhoea. Breast feeding should be allowed as often as the infant desires it.

Breast-fed children are less prone to diarrhoea. Since the human milk has low buffering capacity, stools of breast-fed babies are acidic. Their E. coli count is low, but that of Lactobacillus bifidus is high. Breast milk contains viable phagocytes and other protective substances, such as secretory IgA and specific IgM which protect against most enteropathogens but possibly not against rotavirus infection. Breast-fed children have better growth performance.

2. Non-breast-fed infants: There is some controversy about optimal feeding practices for this group. The general consensus is that the milk should be diluted with an equal volume of water and fed along with ORS till the diarrhoea stops. Thereafter, the child should receive regular formula milk. Meanwhile, *the mother should be reassured that although a temporary increase in the frequency of motions might occur initially, eventual recovery will be faster if the patient continues to be fed.* Some children may have a transient mild disaccharide malabsorption in the

post diarrhoea period as evidence by low pH of stools (less than 5.5, on two occasions) and presence of 1/2 per cent or more of reducing substance in the stools. *This usually does not necessitate withdrawal of milk.* Most patients can tolerate half to two-third diluted milk in such post-diarrhoeal, transient disaccharide malabsorption, which generally lasts 3 to 14 days.

3. **Older infants who are receiving both solid and liquid diet including either breast milk or animal milk:** The diet should contain adequate amount of concentrated foods, so that enough nutrients are absorbed from a small quantity of food. *Milled cereals are preferred to whole cereals.* A well cooked gruel of rice and lentil is usually well tolerated. Mashed bananas are also good. The diet should be iso-osmolar. These foods should be started within 4 to 6 hours of starting the treatment. Soft drinks and fruit juices with high sugar content should preferably be avoided during diarrhoea. Food should be easily digestible and given in smaller quantities at shorter intervals. Contrary to popular belief, most children tolerate *small quantities* of fats and oils which are rich sources of energy, and the diarrhoea does not get worse.

4. **During convalescence:** the dietary intake should be increased to compensate for losses and to promote rapid nutritional recovery.

IX. DRUGS

Most cases of diarrhoea are self-limiting and *generally no medication is necessary except in a few situations.* Drug formulations which enjoy very wide popularity among the physicians include the following:

(i) Antibiotics and chemotherapeutic agents.
(ii) Stool binding agents.
(iii) Drugs reducing motility of the gut.
(iv) Anti-secretory drugs.

Most of these agents have very limited use. Their role is discussed below.

1. ANTIBIOTICS AND CHEMOTHERAPEUTIC AGENTS

Since a large majority of cases of diarrhoea are caused by viruses or toxigenic bacteria and there is little evidence of inflammation of gut mucosa, it is neither necessary nor desirable to use antibacterial substances.

ANTIBIOTICS AND CHEMOTHERAPEUTIC AGENTS FOR DIARRHOEA

Organisms	Antibiotics	Dosage
Shigella	Ampicillin	100 mg/kg in a single dose
	Co-trimoxazole	10 mg TMP plus 50 mg SMX/kg/day in 2 divided doses for 5 days.
	Nalidixic acid	60 mg/kg/day in divided doses for 7 days.
Vibrio cholerae	Tetracycline	25 mg/kg/day in divided doses for 7 days (Do not use in children below 8 years)
	Doxycycline	5mg/kg in a single dose (may be used in younger children)
Enteroinvasive E. coli	As for Shigella	
Campylobacter jejuni	Self-limiting Antibiotics not necessary	Erythromycin, doxycycline, furazolidone, chloramphenicol or aminoglycosides may be used
Non-typhoid Salmonella	Ampicillin	100 mg/kg/day in divided doses for 10 days
Entamoeba histolytica	*Invasive amoebiasis* Metronidazole	35 to 50 mg/kg in 3 divided doses for 4 days
	Luminal amoebicides Diloxanide furoate (Furamide)	20 mg/kg/day in 3 divided doses for 10 days
	Paramomycin	25 to 30 mg/kg/day in 3 divided doses for 5 to 10 days
Giardia lamblia	Metronidazole	20 mg/kg/day in divided doses for 3 days.

Table 4

Antibiotics do not shorten the duration of illness except in cases of cholera. Their indiscriminate use leads to emergence of resistant strains of harmful bacteria and eliminates resident flora which protect the gut. Further more, all drugs are potentially toxic and hazardous. Antimicrobials should be used only for infectious agents such as shigella, cholera vibrio, Entamoeba histolytica and Giardia. They may be prescribed for very small and sick newborn infants infected with enteropathogenic strains of E. coli.

2. BINDING AGENTS

Formulations based on pectin, kaolin or bismuth salts are popular among both physicians and the laity. So far there is little scientific evidence that these are useful. These agents do not reduce excessive losses of fluids and electrolytes, even though the stools appear more solid and the mother is psychologically reassured.

3. ANTI-MOTILITY AGENTS

Synthetic analogues of opiates such as diphenoxylate hydrochloride (Lomotil) and loperamide (Imodium) reduce peristalsis or gut motility. Reduction of gut motility may not abort an attack. On the other hand, *it may give more time for the harmful bacteria to multiply in the gut.* Therefore, the course of illness is often prolonged following their use. These drugs also cause distension of abdomen and other undesirable side effects of opiates. *These are best avoided in infants.* These are rarely used in older children. These may be given judiciously in some cases of chronic diarrhoea and severe tenesmus or cramps.

4. ANTI-SECRETORY AGENTS

Several drugs are currently being evaluated for their anti-secretory properties in the hope that these may reduce the magnitude and duration of diarrhoea and obviate the need for hydration therapy. Such drugs should be safe, inexpensive, capable of oral use and effective against most causes of diarrhoea. Aspirin, chlorpromazine, beta-adrenergic blockers etc. have been evaluated *but are not recommended.*

X. SYMPTOMATIC TREATMENT

1. VOMITING

An occasional vomit in a child need not be treated. In such cases the children can easily tolerate sips of cold water or oral rehydration

solution. If vomiting is persistent, it may be better to delay feeding for a few hours, while at the same time giving clear fluids with a teaspoon or in small sips. One or more doses of metoclopramide 0.1 to 0.2 mg/kg or phenothiazine (0.5 mg/kg) may be given in cases of severe vomiting, but should preferably be avoided since these can cause oculogyric spasms.

2. ABDOMINAL DISTENSION

If bowel sounds are present and the abdominal distension is mild, no specific treatment is necessary. Paralytic ileus due to hypokalemia, necrotising enterocolitis or septicaemia should be suspected if intestinal sounds are absent and distension is gross. In these cases, oral intake should be withheld for some time. Potassium chloride (30-40mEq/L) should be administered intravenously with parenteral fluids if urine is being passed (15 per cent KCI solution provides 2 mEq/ml). The affected child should be screened carefully for any occult infection. Intermittent nasogastric suction gives symptomatic relief.

3. CONVULSIONS

Convulsions associated with diarrhoea may either be due to hypo- or hypernatremia, meningitis, encephalitis, hypocalcemia following bicarbonate therapy for acidosis, cerebral venous thrombosis, or may simply be seizures precipitated by fever. One should also be alert to the possibility of Reye's syndrome, if the preceding vomiting is severe and if there is significant sensorial depression. If hypo-or hypernatremia can be excluded, lumbar puncture must be done. The treatment of convulsions depends on the cause. Symptomatic control of seizures can be achieved either with diazepam (0.2 mg/kg/dose I.V., max. 5 mg), phenobarbitone (5 to 10 mg/kg dose I.M.), paralydehyde (0.1 ml/kg/dose I.M. max. 5 ml) or phenytoin (10 mg/kg/initially I.V. slowly followed by 5 mg/kg/day).

XI. PROTRACTED DIARRHOEA

In about 5 per cent of acute diarrhoea cases in the community, illness may last more than 2 or 3 weeks because of persistent colonisation of upper small intestines by microbes, dietary allergies especially in very young infants and carbohydrate intolerance because of intestinal damage resulting in low levels of disaccharidases. Infants and children with decreased host immunity such as after an attack of measles, or delayed repair of intestinal damage because of associated protein-energy malnutrition are more prone to protracted diarrhoea. *Younger infants who are weaned very early develop intolerance to food proteins such*

CAUSES OF PERSISTENT DIARRHOEA

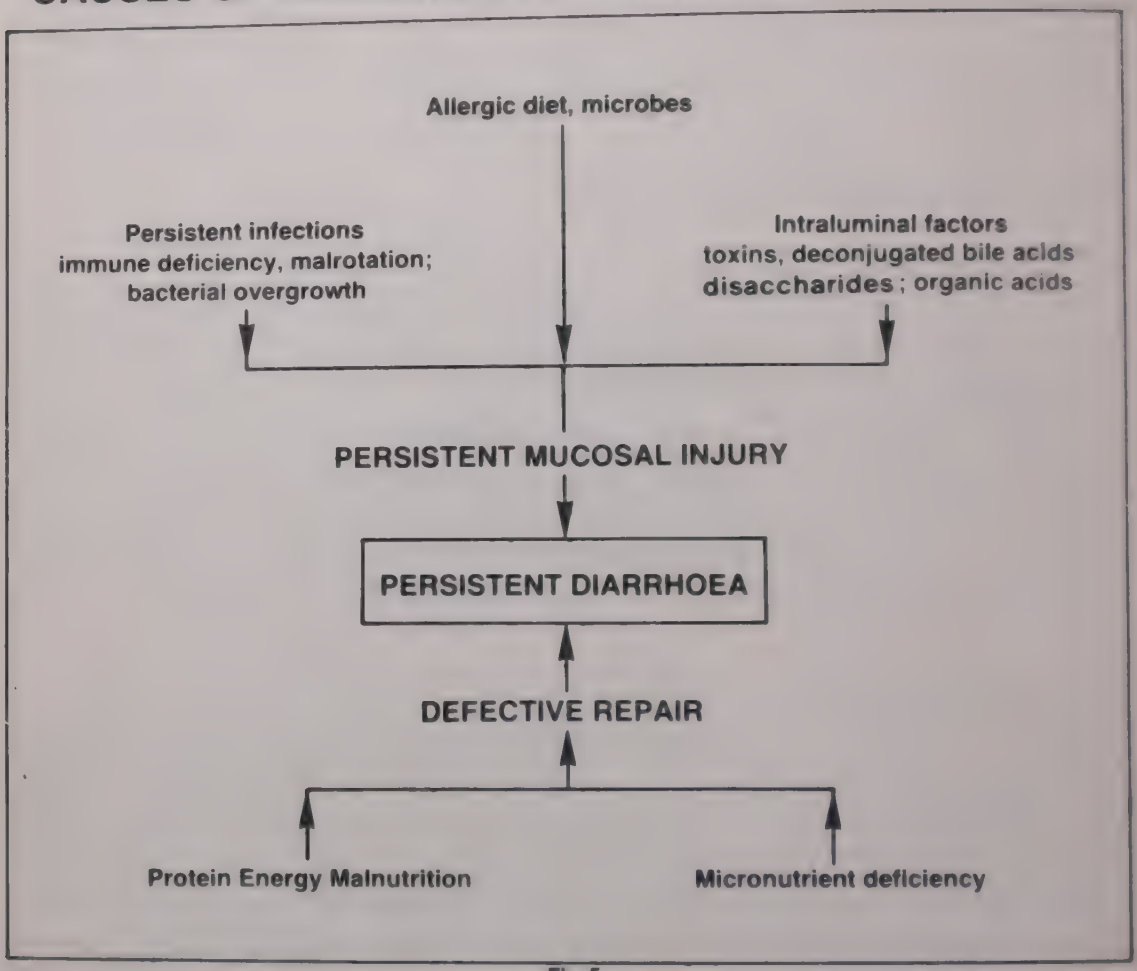


Fig. 5

as cow's milk or even soya milk. Poor personal hygiene and environmental or food contamination may lead to recurrent intestinal infections before the infant recovers from a previous episode. Protozoal infections with Giardia lamblia or Entamoeba histolytica and inadequate treatment of acute diarrhoea are other important causes

In these children, attention should be focused on adequate nutrition intake and selective, judicious use of antimicrobials. Low lactose diets may help when there is carbohydrate intolerance. If the child is given half-diluted milk for a few days with a phased increase in the concentration of milk given in the next week, most cases of protracted diarrhoea improve. Prevention and management of protracted diarrhoea primarily involves encouragement of breast feeding and good nutritional management of acute diarrhoea.

MECHANISM OF PERSISTENT DIARRHOEA

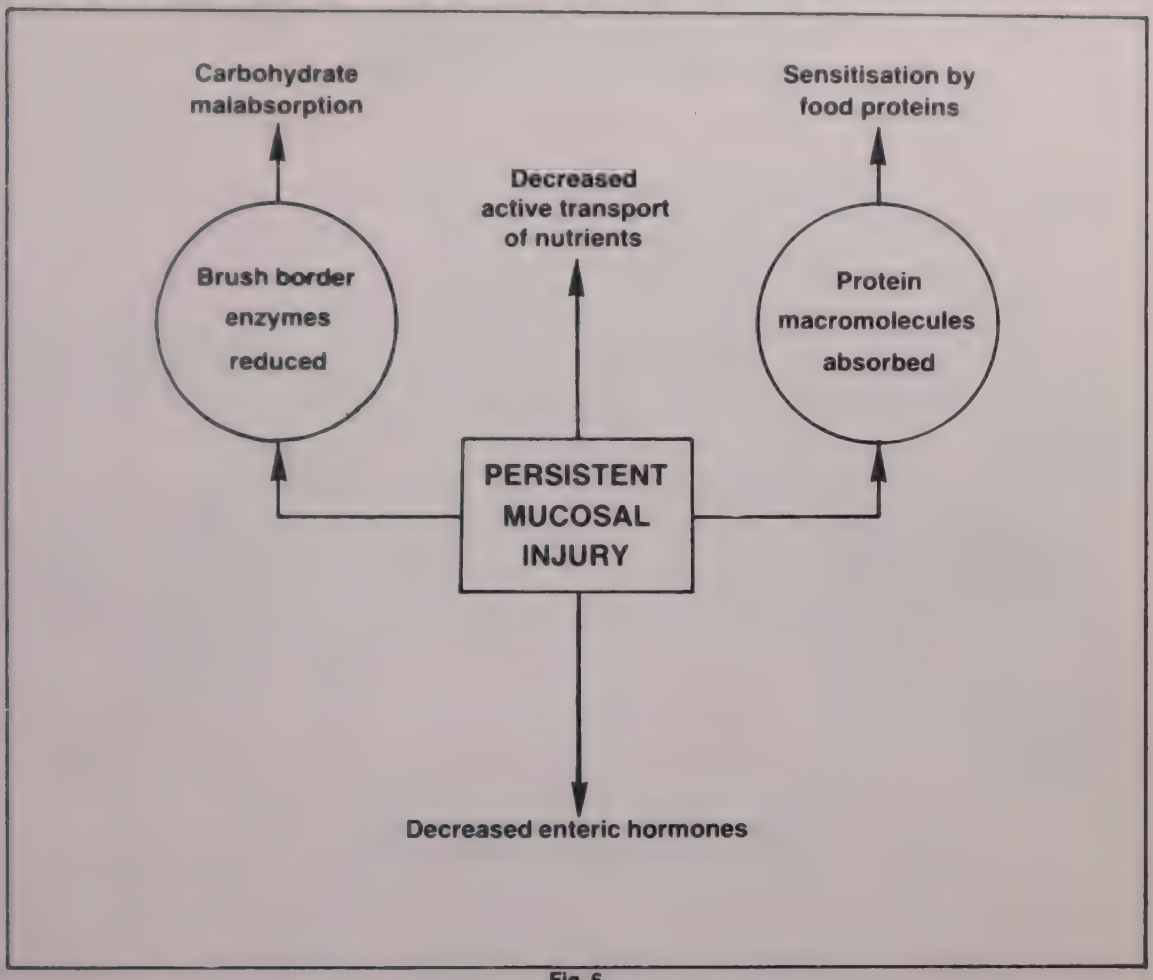


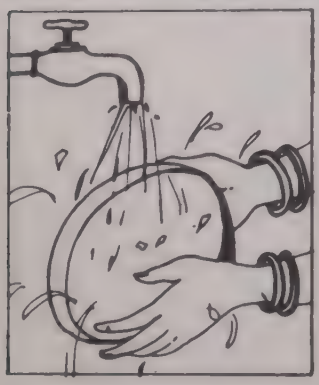
Fig. 6

XII. PREVENTION OF DIARRHOEA AND MALNUTRITION

Prevention of diarrhoea and its nutritional consequences should receive major emphasis in health education. Since breast milk offers distinct advantages and protection from diarrhoea illness, its continuation should be vigorously encouraged and its importance in promoting growth and development of the infant should be stressed. Exclusive breast feeding may not be adequate to sustain growth beyond the first 5 to 6 months of life. Therefore supplementary feeding with energy-rich food mixtures containing adequate amounts of nutrients such as balanced amounts of proteins, fats, iron and vitamins should be introduced by 4 to 5 months of age without stopping breast feeding. Cultural practices such as 'Ann-prashan' ceremony, in which solid

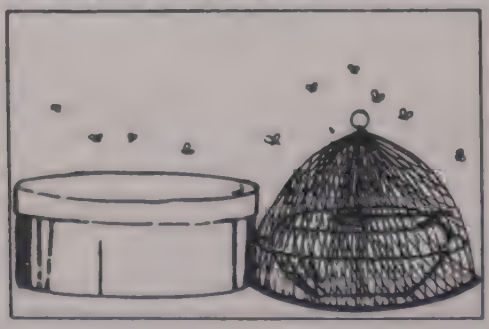
Prevention of Diarrhoea

Clean containers of food



Handwashing before preparing or consuming food

Protect food from dirt, flies, cockroaches



Breast feed babies

Proper sanitation and toilet facilities

Fig. 7

supplements are introduced in the diet at 5 or 6 months of life, should be promoted and reinforced. Complementary feeding should be protected from contamination during preparation,storage or at the time of administration.

Mother should be properly guided to avoid this risk, by concrete recommendations such as use of clean containers, avoiding exposure of food to dust, flies or cockroaches. Proper washing of hands before preparation or administration of food to the baby should be stressed. Water given to the child or used for preparing feeds should be clean, potable, preferably boiled. Vegetables and fruit should be washed and peeled before these are fed to the child. Improvement of environmental sanitation, good water supply, adequate sewage disposal system and protection of food from exposure to bacterial contamination are effective long-term strategies for control of all infectious illnesses including diarrhoea. These measures should be sustained and adequate to achieve the desired goals.